

Review Article

Iodinated Contrast-Enhanced CT and Acute Kidney Injury in the Emergency Department: A Clinical Practice Review

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Abstract: For decades, fear of “contrast-induced nephropathy” has led clinicians to delay or withhold contrast-enhanced computed tomography (CT) in emergency patients whose outcome depended on a rapid diagnosis, based on observational literature that lacked adequate non-contrast control groups. This review synthesises current evidence on the true risk of acute kidney injury (AKI) after intravenous iodinated contrast administration in the emergency department (ED) and proposes a practical, table-based approach for the front-line clinician, drawing on the guidelines of the European Society of Urogenital Radiology, the American College of Radiology and National Kidney Foundation consensus statement, the Kidney Disease: Improving Global Outcomes initiative, and the principal propensity-matched cohort studies and the PRESERVE randomised trial. The most methodologically robust propensity score-matched studies do not show a significant excess risk of AKI, dialysis, or mortality after intravenous contrast-enhanced CT in patients with an estimated glomerular filtration rate (eGFR) of 30 mL/min/1.73 m² or higher, compared with unenhanced CT in matched controls; risk remains uncertain, though probably not negligible, below this threshold or in the presence of established AKI — a reading reinforced by a large contemporary multicentre study finding significantly higher odds of AKI in this range. The term “contrast-induced nephropathy” has consequently given way to the causally more cautious “contrast-associated acute kidney injury” (CA-AKI). The PRESERVE trial showed no benefit of sodium bicarbonate over isotonic saline, nor of N-acetylcysteine over placebo, in patients at high cardiovascular risk undergoing angiography. In the unstable patient, or when the diagnosis under consideration is life- or organ-threatening, contrast administration should almost never be delayed to “wait for creatinine.” Rational prevention rests on targeted risk stratification, simple hydration in stable high-risk patients, and avoidance of redundant contrast exposures, rather than on pharmacological prophylaxis protocols whose efficacy has not withstood randomised testing.

Keywords: Contrast-Associated Acute Kidney Injury (CA-AKI); Intravenous Iodinated Contrast; Contrast-Enhanced Computed Tomography (CT); Acute Kidney Injury (AKI); Emergency Department; Estimated Glomerular Filtration Rate (eGFR).

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INTRODUCTION AND BACKGROUND

Intravenous iodinated contrast material is integral to most CT examinations performed in emergency medicine: sepsis work-up, polytrauma assessment, CT angiography for suspected large-vessel occlusion stroke, CT pulmonary angiography for suspected pulmonary embolism, and CT angiography for suspected aortic syndromes. Since the 1980s, the radiological literature has propagated the notion that contrast material could, by itself, cause acute kidney injury — so-called contrast-induced nephropathy. This belief became deeply embedded in clinical practice and

has long justified diagnostic delay, substitution of less sensitive unenhanced protocols, or outright refusal of contrast administration in patients whose clinical state demanded rapid imaging.

Over the past decade, a series of methodologically stronger studies — using genuine non-contrast control groups and propensity score matching — has substantially undermined this paradigm. Most cases historically attributed to contrast nephrotoxicity appear to represent multifactorial AKI (hypovolaemia, sepsis, concurrent nephrotoxins, haemodynamic instability), in

which contrast exposure was a coincidental confounder rather than a causal agent [1, 2].

This review gives the emergency physician an updated, guideline-based framework for deciding when contrast can be given without delay, when caution is warranted, and how to organise prevention and monitoring without sacrificing the diagnostic speed that defines emergency medicine.

It is a narrative review; sources were identified through PubMed/MEDLINE, Radiology, the New England Journal of Medicine, and Kidney International (January 2012–July 2026), supplemented by hand-searching the reference lists of major society guidelines, with priority given to randomised trials, propensity-matched cohort studies, systematic reviews/meta-analyses, and current guidelines over single-centre observational data or expert opinion where both were available. A 2026 KDIGO AKI/AKD guideline was available as a public-review draft at the time of preparation; because it had not yet reached final publication, its draft recommendations were not presented as finalised guideline recommendations.

REVIEW

1. From Contrast-Induced AKI to Contrast-Associated AKI: A Paradigm Shift

The terminological shift is not cosmetic; it reflects a reappraisal of causality itself. “Contrast-induced nephropathy” (CIN) and “contrast-induced acute kidney injury” (CI-AKI) are historical terms implying a direct cause-and-effect relationship between contrast injection and renal decline. “Contrast-associated acute kidney injury” (CA-AKI) is the term now favoured by the ACR-National Kidney Foundation consensus and the higher-quality literature, describing a temporal association without presupposing causation — AKI occurring after contrast exposure, whose attribution to the contrast agent itself must be established case by case rather than assumed [7-9].

The studies that drove this shift share a common methodology: comparing contrast-exposed patients with unexposed patients of comparable risk through propensity score matching, rather than tracking creatinine after contrast without a control arm. A landmark study stratified by baseline eGFR found no significant contrast-attributable excess risk of AKI, including in the intermediate eGFR stratum (30–59 mL/min/1.73 m²) [3, 4]. A separate large single-centre matched cohort of 21,346 patients found that contrast exposure was not an independent risk factor for emergent dialysis or short-term mortality, even among patients with comorbidities historically thought to predispose to nephrotoxicity [5]. A meta-analysis of 21 propensity-matched cohorts reached a concordant conclusion: intravenous contrast was not associated with a significant excess of AKI, dialysis, or mortality in the general CT population, including at eGFR of 45 mL/min/1.73 m² or higher [6].

This does not mean contrast nephrotoxicity has been disproven altogether — it remains documented in interventional imaging with high-dose intra-arterial administration proximal to the renal circulation — but the risk associated with diagnostic intravenous CT, at current low-osmolality doses, has likely been substantially overestimated for decades [1-6].

2. How large is the Actual Risk in Emergency Patients?

CA-AKI is substantially more common than true CI-AKI because CA-AKI includes AKI from any cause occurring after contrast exposure. In the ACR–NKF consensus, estimated CA-AKI rates after contrast-enhanced CT were approximately 5% at eGFR ≥60, 10% at eGFR 45–59, 15% at eGFR 30–44, and 30% at eGFR <30 mL/min/1.73 m². By contrast, the estimated risk of CI-AKI was near 0% at eGFR ≥45, 0–2% at eGFR 30–44, and 0–17% at eGFR <30 mL/min/1.73 m², with substantial uncertainty in the latter group [7]. These estimates should not be interpreted as ED-specific causal risks; dedicated ED cohorts confirm that CA-AKI remains associated with baseline kidney disease and other acute or chronic comorbidity [10, 11].

Table: CI-AKI risk by baseline eGFR

Baseline eGFR (mL/min/1.73 m ²)	Approximate CA-AKI incidence	Estimated CI-AKI risk	Interpretation
≥60	≈5%	Not reliably measurable; likely very low	CA-AKI may occur; most events are not necessarily caused by contrast
45–59	≈10%	Near 0%	Low incremental CI-AKI risk; proceed when clinically indicated
30–44	≈15%	0–2%	CA-AKI is more frequent; individualise risk assessment
<30	≈30%	0–17%, uncertain	Highest uncertainty; severe CKD/AKI warrants individualised assessment

This uncertainty below an eGFR of 30 is not merely a gap in older data — it is actively reinforced by

more recent, well-powered studies pointing in different directions. A 2023 multicentre analysis of 75,328

patients found significantly higher odds of CI-AKI at an eGFR of 30–44 (odds ratio 1.34) and below 30 (odds ratio 1.78) compared with unenhanced CT, with even higher odds in diabetic patients with severely reduced eGFR [12]. An ED-specific propensity score-matched analysis similarly found a higher adjusted risk of AKI in patients with chronic kidney disease receiving contrast (adjusted risk ratio 1.60), although downstream dialysis initiation remained rare in both the contrast and non-contrast groups (0.7% versus 0.3%) [13]. These contemporary, adequately powered studies argue against a categorical claim of safety below an eGFR of 30, and support the individualised, case-by-case approach recommended throughout this review rather than a blanket reassurance.

The key clinical distinction is therefore between the relatively frequent occurrence of CA-AKI and the much smaller, genuinely contested incremental risk of CI-AKI. The highest uncertainty remains in patients with eGFR <30 mL/min/1.73 m² and in those with active AKI, while diagnostic delay may itself cause substantial harm [7, 14].

3. Which Emergency Patients Are Truly at Risk?

Modern risk stratification should therefore combine kidney function, presence of AKI, hemodynamic status, volume status, and the urgency of the diagnostic indication rather than rely on a single universal eGFR threshold, an approach echoed by ED-specific risk-scoring frameworks that combine clinical and laboratory parameters beyond eGFR alone [7-15].

Table 1: Clinical factors associated with increased concern for CA-AKI in emergency patients

Risk factor	Relative importance	Practical interpretation
Active AKI	Very high	Major marker of renal vulnerability; individualized decision
eGFR < 30 mL/min/1.73 m ²	High	High uncertainty about incremental CI-AKI risk; weigh benefit vs risk
CKD stage 4–5	High	High-risk context; consider prophylaxis when appropriate
Severe hypovolaemia	High	Correct clinically important volume depletion
Shock / hypotension	High	Treat shock; do not delay vital imaging solely for renal protection
Sepsis	Moderate–high	Independent AKI driver; contrast is not an automatic contraindication
eGFR 30–44 mL/min/1.73 m ²	Moderate	Usually proceed when indicated; consider prophylaxis only in selected high-risk patients
Heart failure / volume overload	Moderate	Balance any hydration strategy against volume-overload risk
Advanced age	Low–moderate	Contextual marker; not an independent contraindication
Diabetes mellitus	Low–moderate	Does not by itself justify a different eGFR threshold; consider underlying CKD
Concurrent nephrotoxic drugs	Context-dependent	Avoid nonessential nephrotoxins when feasible, particularly in high-risk patients
Recent contrast exposure	Context-dependent	Consider cumulative clinical context; avoid unnecessary repeat exposure when feasible

A patient without known kidney disease or other major risk factors generally requires no specific prophylactic measure before intravenous iodinated contrast administration. This does not remove the need to assess clinically apparent hypovolemia, shock, or other causes of AKI.

4. Should Renal Function be assessed before Contrast Administration?

Current ACR guidance supports risk-based rather than universal pre-contrast kidney-function

assessment before intravenous iodinated contrast. Routine creatinine/eGFR measurement is not required for patients without identified risk factors; targeted assessment is appropriate when kidney disease, prior AKI, diabetes or other relevant risk factors are present, provided that testing does not delay time-critical imaging [7-16].

Table 2: Pre-contrast renal assessment in the emergency department

Level	Clinical picture	Action
1 — No renal risk identified	Young, no renal history, no diabetes, stable	No routine creatinine required solely for contrast; proceed when clinically indicated
2 — Possible renal vulnerability	Known CKD, diabetes, treated hypertension, nephrotoxic drugs, prior renal transplant/solitary kidney	Obtain creatinine/eGFR when readily available, but do not delay a time-critical examination

Level	Clinical picture	Action
3 — Known/suspected major renal dysfunction	eGFR < 30 mL/min/1.73 m ² , active AKI, dialysis-dependent	Assess urgency; individualized discussion for deferrable studies; severe CKD/AKI is not an automatic contraindication

In the acutely ill patient, serum creatinine may lag behind the onset of AKI, and an eGFR calculated from a non-steady-state creatinine should be interpreted cautiously. Nevertheless, absence of a recent creatinine value should not by itself delay imaging when the indication is time-critical [7-16].

5. Should Contrast-Enhanced CT be delayed in Patients with AKI or Reduced Egfr?

When the indication is vital or time-critical, contrast-enhanced imaging should generally proceed without waiting for renal laboratory results if the result will change immediate management. This includes suspected acute aortic syndrome, large-vessel occlusion stroke, major trauma, high-risk pulmonary embolism, and suspected mesenteric ischemia. The decision should

be based on the overall clinical benefit-risk balance; severe CKD or AKI increases uncertainty about CI-AKI risk but is not, by itself, an automatic contraindication [7-16].

When the indication is non-vital and deferrable, eGFR <30 mL/min/1.73 m² or active AKI warrants individualized assessment and, where appropriate, discussion with the radiologist and/or nephrologist. A noncontrast alternative or optimization of reversible factors may be considered. The ACR–NKF consensus treats severe kidney dysfunction or recent AKI as a high-risk context rather than an automatic contraindication [7].

6. Prevention of CA-AKI in the ED

Table 3: Prevention of CA-AKI in emergency patients

Measure	Recommendation
Correct modifiable insults	Correct clinically important hypovolemia, hypotension and shock independently of the contrast decision
IV isotonic fluids	Routine prophylaxis is not required for stable eGFR ≥30; IV saline is preferred when prophylaxis is indicated for AKI or eGFR < 30, if not contraindicated
Saline vs sodium bicarbonate	No clinically important superiority of bicarbonate over isotonic saline; saline is preferred when prophylaxis is indicated [17]
N-acetylcysteine	Not recommended for routine prophylaxis [17]
Statins / other pharmacological agents	No established role as routine ED prophylaxis
Nephrotoxic drugs	Withhold nonessential nephrotoxins when feasible in high-risk patients; do not compromise necessary treatment
Metformin	For IV iodinated contrast: continue if eGFR ≥30 and no AKI; withhold in AKI or eGFR < 30; follow ESUR/local policy for intra-arterial first-pass exposure [16, 18]
Contrast agent and dose	Use low- or iso-osmolar agents; use the lowest dose that maintains diagnostic quality
Repeated administration	Avoid unnecessary repeat injections, particularly in high-risk patients; emergency diagnostic need takes precedence
Patients at risk of fluid overload	Avoid protocolised aggressive hydration in heart failure or shock; tailor to haemodynamic status

The PRESERVE trial demonstrated no benefit of sodium bicarbonate over isotonic saline, nor of N-acetylcysteine over placebo, in a high-risk angiography population [17], an accompanying editorial cautioned specifically against uncritically extrapolating these results to lower-risk diagnostic imaging populations, for which no equivalently powered trial exists [19]. There is no justification for delaying vital imaging solely to

complete a prophylactic hydration protocol. In high-risk patients, correction of clinically important hypovolemia and other hemodynamic abnormalities should proceed in parallel with, rather than instead of, necessary imaging [7-18].

7. Special Emergency Scenarios

Table 4: Time-critical emergency scenarios and contrast administration

Scenario	Approach
Sepsis / septic shock	Contrast-enhanced imaging for source identification should not be delayed solely for renal testing or prophylaxis when time-critical
Major trauma	Use contrast-enhanced CT when clinically indicated; immediate hemorrhage detection takes priority over uncertain incremental CI-AKI risk

Scenario	Approach
Acute ischaemic stroke	Do not routinely await creatinine before CTA/CTP when large-vessel occlusion is suspected and imaging will guide urgent reperfusion therapy
Pulmonary embolism	CT pulmonary angiography remains standard when indicated; V/Q imaging is an alternative when clinically appropriate
Acute aortic syndromes	Do not delay emergency CTA when acute aortic syndrome is suspected and imaging is required for immediate management
Acute mesenteric ischaemia	Proceed with contrast-enhanced CT when mesenteric ischemia is suspected and imaging is needed for urgent diagnosis

8. Post-Contrast Monitoring

Table 5: Post-contrast monitoring according to clinical risk

Risk category	Monitoring
Low risk (no identified risk factor)	No routine post-contrast creatinine monitoring solely because contrast was administered
Identified risk factor (eGFR < 30–45 mL/min/1.73 m ² , pre-existing AKI, cumulative risk factors, hospitalised patient)	Repeat creatinine when clinically indicated or as part of routine inpatient monitoring; consider 48–72 h in substantial-risk patients
Confirmed creatinine rise	Search actively for alternative causes of AKI before attributing the rise to contrast

The diagnosis of AKI should follow KDIGO criteria: an increase in serum creatinine by ≥ 0.3 mg/dL (≥ 26.5 $\mu\text{mol/L}$) within 48 hours, or to ≥ 1.5 times baseline within the preceding 7 days, or urine output < 0.5 mL/kg/h for 6 hours. A temporal creatinine rise after contrast exposure should be termed CA-AKI unless causality can be established [22].

9. A Practical ED Algorithm

Step 1: Is the indication vital or time-critical? (e.g., acute aortic syndrome, stroke potentially eligible for thrombectomy, major trauma, severe sepsis/shock without a source, high-risk pulmonary embolism, or mesenteric ischemia). If yes: do not delay necessary contrast-enhanced imaging solely to obtain creatinine; arrange renal monitoring afterward when clinically indicated. If no: proceed to step 2.

Step 2: Does the patient have a clinically relevant kidney-risk factor? (known CKD, prior AKI, suspected current AKI, selected nephrotoxic exposures, or other substantial renal vulnerability). Diabetes alone should not automatically impose a different eGFR threshold. If no major risk factor is identified, routine pre-contrast creatinine is generally unnecessary [7-16].

Step 3: If kidney function is clinically relevant and can be obtained promptly, assess creatinine/eGFR without unduly delaying the examination. eGFR ≥ 30 mL/min/1.73 m² without active AKI: proceed when clinically indicated; routine prophylaxis is not required. eGFR 30–44 with multiple risk factors or recent AKI: consider individualized prophylaxis. eGFR < 30 mL/min/1.73 m² or active AKI with a deferrable indication: individualize the decision and consider alternatives or optimization of reversible factors. eGFR < 30 mL/min/1.73 m² or active AKI with a non-deferrable

indication: proceed when the diagnostic benefit is compelling, documenting the risk-benefit rationale [7-18].

Step 4: In every case: use a contrast dose sufficient for diagnostic quality without arbitrary dose reduction below a validated diagnostic threshold; use modern low- or iso-osmolar agents; avoid unnecessary closely spaced repeat injections; correct clinically important hypovolemia; and manage metformin according to current ACR/ESUR recommendations [7-18].

10. Controversies and Common Misconceptions

Whether clinically meaningful CI-AKI occurs after modern intravenous CT remains uncertain, particularly in patients with eGFR < 30 mL/min/1.73 m² or active AKI. The strongest controlled observational evidence does not demonstrate excess AKI, dialysis, or mortality attributable to intravenous contrast in most patients, whereas severe kidney dysfunction remains under-represented in these same cohorts and therefore genuinely uncertain [1-7]. This under-representation is a structural methodological limitation rather than a minor caveat: patients with the most severe renal impairment are systematically less likely to be selected for contrast administration in the first place, which limits the power of matched-cohort designs to detect a true effect specifically where it would matter most [20]. This is precisely the population in which the Lee *et al.*, and Kene *et al.*, findings [12, 13], diverge from the larger, lower-risk-dominated matched cohorts — underscoring that this remains an open question rather than a settled one, a position increasingly echoed in contemporary imaging guidance moving away from rigid, single-threshold restrictions toward individualised, indication-driven decision-making [21].

Table 6: Common misconceptions regarding iodinated contrast in the ED

Common belief	Contemporary interpretation
Elevated creatinine automatically means no contrast	Incorrect; assess kidney function, AKI status and clinical urgency
eGFR < 30 is an absolute contraindication	Incorrect; high-risk context requiring individualized assessment, not an automatic contraindication
Any AKI after contrast proves contrast nephrotoxicity	Incorrect; CA-AKI is temporal association, whereas CI-AKI requires causal attribution
Every at-risk patient needs prophylactic IV fluids	Incorrect; prophylaxis is targeted to selected high-risk patients
N-acetylcysteine prevents CA-AKI	Not supported by randomized evidence for routine prophylaxis [17]
Bicarbonate is superior to saline	No clinically important superiority over isotonic saline [17]
Metformin must always be stopped before contrast	Incorrect; management depends on eGFR, AKI and route of contrast administration [16, 18]
Creatinine must be available before every contrast CT	Incorrect; use targeted, risk-based assessment [7, 16]
Avoiding contrast is always the “renal-safe” choice	Incorrect; overall patient safety and diagnostic urgency should guide the decision

CONCLUSIONS

The paradigm surrounding contrast-associated kidney injury has shifted substantially. Higher-quality evidence indicates that intravenous iodinated contrast is not an important independent cause of AKI for most patients, while the incremental risk remains genuinely contested — not merely under-studied — in those with severe kidney dysfunction or active AKI, as recent large studies reach conflicting conclusions in this specific subgroup [7-13]. For emergency physicians, the practical message is to avoid unnecessary diagnostic delay, use targeted rather than universal renal assessment, correct clinically important reversible risk factors, and avoid ineffective pharmacological prophylaxis. The decision to administer contrast should integrate the urgency and diagnostic value of imaging with the patient’s overall renal and hemodynamic risk [7-18].

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